IJPMR 7, April 1996

Role of Motor Nerve Conduction Velocity and 'H' Reflex & 'F' wave Latency in diabetic Neuropathy

Dr. S. Ramar

Abstract

A study on role of "Motor nerve conduction velocity and 'H' reflex & "F" wave latency" was undertaken to evole the relative merits of the said methods by EMG study for early detection of subclinical neuropathy. IDDM and NIDDM including newly diagnosed diabetics were included along with age matched control cases. The observations revealed that (1) subclinical neuropathy in diabetes mellitus can be identified by motor nerve conduction velocity of all the peripheral nerves, and "H" reflex & "F" wave latency, (2) the delay in motor nerve conduction is much pronounced in distal segment than proximal segment of ulnar and median nerves, (3) the delay in motor conduction was greater in lower limbs than upper limbs, (4) IDDM patients exhibited statistically significant reduction in motor conduction velocity in the proximal segment of unlar nerve and distal segment of median nerve the NIDDM, (5) newly diagnosed diabetics showed significant delay in motor nerve conduction of all the peripheral nerves except terminal latency of tibial nerve, and "H" reflex & "F" wave latency, (6) "H" reflex & "F" wave latency evinced good correlation with severity of diabetes.

Key words : MNCV = Motor Nerve Conduction Velocity; IDDM = Insulin Dependent Diabetes Mellitus; NIDDM = Non Insulin Dependent Diabetes Mellitus.

Introduction

The prevalence of diabetes mellitus is variable in the global population. The prevalence of diabetes mellitus in India is about 9%. The rising prevalence of diabetes mellitus from 2.5% to about 9% in urban India is related to the replacement of traditional cooking oil such as ghee, cocount oil, mustard oil containing low ratio of free radicals viz., omega 6 and omega 3 fatty acids with polyunsaturated fatty acids such as sunflower and safflower oil containing high ratio of free radicals and reduced intake of antioxidants - vitamin "C" and vitamin "E". The prevalence of diabetes in rural India is unchanging and is around 2.5%. This wide difference in prevalence of diabetes mellitus in rural India is due to the habit of cooking with traditional cooking oil containing low ratio of omega 6 and omega 3 fatty acids[1]. In 1990 it was estimated that there were 15 million people with diabetes in India. It may rise to 35 million by the year 2000. This hiking prevalence of diabetes mellitus demands improved strategy to curb and control diabetes mellitus.

The commonest complication of diabetes mellitus is polyneuropathy. The diabetic polyneuropathy is due to metabolic derangement caused by chronic hyperglycemia[2]. Poor glycemic control is an essential permissive factor in the early development of diabetic neuropathy[3]. In the early phase of diabetic polyneuropathy there is a functional rather than structural damage of the nerve[4]. Hence early detection of polyneuropathy

Correspondence : Dr. S. Ramar Department of Physical Medicine and Rehabilitation. Tirunelveli Medical College Hospital Tirunelveli- 627011 Tamilnadu - India

during the subclinical stage can permit reversal of pathological process by good glycemic control.

The electrophysiological studies of peripheral nerves viz., motor nerve conduction velocity, sensory nerve conduction velocity, "H" reflex study, "F" wave study, distribution of motor nerve conduction velocity using nerve impulse collision method and single fibre EMG are used to evaluate peripheral neuropathy both during its subclinical and clinical phases of neuropathy both during its subclinical and clincial phases of neuropathy. Nerve conduction velocity of dorsal nerve of penis and bulbocavernous reflex are performed to assess the diabetic impotence. Visual evoked potential study are useful to assess diabetic optic neuropathy. Each method of electrophysiological study has its own merits and demerits.

The study on motor nerve conduction velocity of median, ulnar, popliteal and tibial nerves; "H" reflex and "F" wave study were undertaken to assess its significance for early detection of subclinical neuropathy because of its simplicity.

Method

The criteria laid down by WHO expert group 1980 and American Diabetic Association[5]: (1) in nonpregnant adult patients with classic symptoms such as polyuria, polydispsia, rapid loss of weight and random blood sugar more than 200mg % (or) (2) in asymptomatic patients above value is certainly adequate as well found more than one occasion (or) (3) if hyperglcemia is not marked but fasting blood sugar more than 120 mg% (7 m.mols/litre) and post glucose level (done 2 hours after taking 75 gms of oral glucose) more than 200 mg% 10 m.mols/litre) were used to select 50 cases each for the study on motor nerve conduction velocity and "H" reflex & "F" wave latency. A 20 age matched controls were included in the study on "Motor nerve conduction velocity" after excluding diabetes mellitus, peripheral neuropathy of any origin and factors triggering peripheral neuropathy. Similarly 25 age matched cotrols were included in the study on "H" reflex and "F" wave latency.

Operational Definition

The cases under study were classified into diabetes with clinical neuropathy and those without it based on the clinical parameters: (1) history of paresthesia (2) impaired (or) absent pain sensation (3) dull (or) absent vibration sensation (4) diminished (or) absent postural sensation (5) dull (or) absent ankle jerk.

The duration of diabetes mellitus was calculated in months from the onset of symptoms (or) from the time of detection whichever may be earlier. The diabetes mellitus was also divided into IDDM and NIDDM. The severity of diabetes mellitus was graded into I if fasting blood sugar is below 150% mg%, II if fasting blood sugar is between 150 mg% and 200 mg% and III if fasting blood sugar is above 200 mg%. Newly diagnosed diabetes mellitus patients were also included.

EMG study was done by using ECIL MDM 30 two channel Storage Myograph. Motor nerve conduction velocity of all the peripheral nerves viz., median, unlar, peroneal and tibial nerves were studied by using silver disc surface electrodes kept at 3 cm apart over the distal most accessible muscle[6]. The ground electrode was applied between stimulating and recording electrodes. In motor nerve conduction velocity study, the median nerve and unlar nerve were stimulated at axilla, elbow and wrist and the compound motor action potentials were recorded from the abductor pollicis brevis for median nerve and abductor digiti minimi for unlar nerve. The peroneal nerve was stimulated at the level of neck of fibula and at flexor retinaculum behind the medial malleolus and the compound motor action potentials were recorded from abductor hallucis longus. Proximal, distal and terminal latencies and segmental length of all the nerves were recorded for computing the motor nerve conduction velocity.

The "H" reflex study was done by stimulating the tibial nerve at the popliteal fossa. Supramaximal stimulus was used to elicit "M" response. Its latency and amplitude was measured. The latency of "M" response represents the time taken for the impulse to travel from the popliteal fossa distally to the soleus muscle. Subsequently submaximal stimulus was used to stimulate selectively group I A fibres to elicit "H" reflex. Its latency represents the time taken for the impulse to travel from the popliteal fossa proximally to the spinal cord and then to the soleus muscle through the alpha fibres[7]. "F" wave was elicited by stimulating the tibial nerve in the popliteal fossa. It was recorded in the soleus muscle using supra maximal stimulation. When supramaximal stimulus was used to elicit "M" response, some of the impulses travel antidromically i.e., proximally to the first node of Ranvier and reflected back distally along the same motor fibres to the soleus muscle. This explains the delayed response. "F" wave amplitude is usually below 200 microvolts in contrast to higher amplitude of "H" response which is as high as 10,000 micro volts. The frequency of "F" response is one per ten antidromic stimuli where as "H" response is elicited for every stimulus.

Enumeration and percentage were used for the discussion of clinical data. "t" tests and Pearson's correlation coefficient were claculated to assess the level of association of various electromyographic readings. The mean value of motor nerve conduction velocity, "H" reflex latency and "F" wave latency were taken for correlation of severity and duration of diabetes.

EMG readings of individual nerves were taken for comparision with control in 't' tests.

Results

Among the 50 cases of diabetes mellitus included in the group on motor nerve conduction velocity, 24% were between 10-29 years of age, 20% were between 30-49 years of age abd 56% were above 50 year of age. Among the 50 cases included under "H" reflex and "F" wave latency study, 18% were between 10-25 years of age, 30% were between 30-49 ears of age and 42% were above 50 years of age. The prevalence of diabetes showed male preponderence i.e., 80% and 72% respectively in the study on motor nerve conduction velocity and "H" reflex & "F" wave latency. IDDM constitutes 32% of population in the group on motor nerve conduction velocity study and 24% in the group on "H" reflex & "F" wave latency study. NIDDM constitutes 68% in the group on motor nerve conduction velocity study and 76% in the group on "H" reflex & "F" wave latency study. Patients with clinical evidence of peripheral neuropathy forms 56% in the group on motor nerve conduction velocity and 48% in the group on "H" reflex & "F" wave latency study. Patients with grade I diabetes constitutes 6% in the group on motor nerve conduction velocity and 22% in the group on "H" reflex & "F" wave latency; grade II diabetes, 16% in the group on motor nerve conduction velocity, 32% in the group on "H" reflex & wave latency: and grade III diabetes, 68% in the group on motor nerve conduction velocity and 46% in the group on "H" reflex & "F" wave latency.

There was significant delay in motor nerve conduction velocity of all the peripheral nerves studie in both groups with and without clinical neuropathy. Nerve conduction delay was much pronounced in distal segments than in proximal segments of upper limb. The delay in motor nerve conduction velocity was greater in lower limb than upper limb (Table-1).

Table 1

Motor Nerve Conduction VelocityControl Vs Diabetes Mellitus

Nerve Studied	Control Metres/second	With Neuropathy Metres/second	Without Neuropathy Metres/second
		*****	*****
Peroneal	51.04 ± 5.08	41.41 ± 6.65	41.15 ± 6.39
		*****	*****
Tibial	50.01 ± 4.19	40.23 ± 5.91	39.57 ± 5.88
		*****	*****
Median			
Axilla to Wrist	63.03 ± 4.20	54.93 ± 4.07	53.22 ± 5.09
Axilla to elbow	69.47 ± 10.38	62.98 ± 9.84	64.23 ± 11.19
		*****	*****
Elbow to Wrist	59.26 ± 6.77	49.09 ± 6.11	45.20 ± 5.92
		*****	*****
Ulnar			
Axilla to Wrist	60.65 ± 5.24	52.22 ± 5.37	51.25 ± 5.67
		*****	***
Axilla to elbow	60.77 ± 7.00	55.98 ± 8.78	53.50 ± 7.98
		*****	*****
Elbow to Wrist	60.53 ± 6.43	49.65 ± 7.03	50.20 ± 6.55

Significant at * - p less than 0.05 *** p less than 0.01, ***** - p less than 0.0001.

Terminal latency in all the peripheral nerves were also prolonged both in the group with and without neuropathy except in tibial nerve in the group without neuropathy (Table-2) There was a relative reduction in motor nerve conduction velocity in IDDM than in NIDDM with a statistically significant reduction in proximal segment of ulnar nerve and distal segment of median nerve (Table-3). The delay in motor nerve conduction velocity was also oberved in newly diagnosed diabetics with reference to control cases with significant "P" value in all the peripheral nerves. Similarly the terminal latency was also prolonged in newly diagnosed diabetics with significant "P" value except in tibial nerve (Table-4). The duration and severity of diabetes mellitus did not show statistically signficant correlation with motor nerve conduction velocity.

"H" reflex latency was prolonged in both group with and without clinical neuropathy. Similar delay in motor nerve conduction velocity of tibial nerve was observed in both groups with and without clinical neuropathy. Where as statistically significant delay was observed in "F" wave latency study only in the group with clinical neuropathy (Table-5) "H" reflex reflex latency was prolonged both in IDDM and NIDDM without any significant difference between them. Tibial nerve conduction velocity and "F" wave latency showed statistically significant delay in both IDDM and NIDDM (Table-6) Newly diagnosed diabetes also showed a statistically significant delay in all the three parameters viz. "H" reflex, "F" wave latency and tibial nerve conduction velocity (Table-7). Age of the patient and duration of diabetes did not show any correlation with "H" reflex latency, "H" wave latency and tibial nerve conduction velocity. However severity of diabetes showed correlation with "H" reflex latency and "F" wave latency but not with tibial nerves conduction velocity (Table-8,9,10)

Discussion

The prevalence of diabetes mellitus in this study showed usual male preponderehce. Though the incidence of diabetes mellitus is more after 50

Table 2

Terminal latency

Control Vs Diabetes Mellites

Nerve Studied	Control Metres/Second	With Neuropathy Metres/Second	Without Neuropathy Metres/second
Peroneal	4.44 ± 0.60	4.75 ± 0.76 *	5.18 ± 1.03 *
Tibial	4.63 ± 0.73	4.74 ± 0.76 ****	5.03 ± 1.07 ***
Median	3.65 ± 0.40	4.02 ± 0.71	4.22 ± 0.56
Ulnar	2.92 ± 0.42	3.25 ± 0.56	3.43 ± 0.74

Significant at * - p less than 0.05, **** p less than 0.001, ***** - p less than 0.0001.

Table 3

IDDM Vs NIDDM - Motor nerve conduction velocity

Types of Diabetes	Ulnar nerve Proximal	Ulnar nerve Distal	Median nerve Proximal	Median nerve Distal	Tibial nerve	Peroneal nerve
IDDM n-16	53.1875 ± 9.53	46.3125 ± 5.74	58.1875 ± 6.83	47.875 ± 6.45	40.4375 ± 14.55	38.125 ± 7.16
NIDDM n-34	65.3529 ± 7.86 ***	47.5588 ± 4.81	55.9118 ± 5.18	51.4412 ± 5.18 *	42.7059 ± 4.98	41.2353 ± 4.82
"t"	4.767	0.8030	1.3061	2.0969	0.8203	1.8123

Significant at * - p less than 0.05, ** p less than 0.02, *** p less than 0.01, ***** - p less than 0.0001.

Table 4

Motor Nerve Conduction Velcoity
Control Vs Newly diagnosed Diabetes Mellitus

Nerve Studied	Control Metres/Second	Newly diagnosed Diabetics Metres/second
Peroneal	51.04 ± 5.08	39.42 ± 3.42 *****
Tibial	50.01 ± 4.19	39.71 ± 3.19 *****
Median		
Axilla to Wrist	63.03 ± 4.20	53.77 ± 3.50 *****
Axilla to elbow	69.47 ± 10.38	56.40 ± 6.50 *****
Elbow to Wrist	59.26 ± 6.77	51.14 ± 3.53 *****
Ulnar		
Axilla to Wrist	60.65 ± 5.235	49.65 ± 4/35
Axilla to elbow	60.77 ± 7.026	55.41 ± 5.90 *****
Elbow to Wrist	60.53 ± 6.43	46.00 ± 5.79

Significant at * - p less than 0.05, *** p less than 0.01, ***** p less than 0.0001

Table 5

'H' Reflex & 'F' wave latency - control Vs Diabetes Mellitus

EMG Study	Control (1)	With Neuropathy (2)	Without Neuropathy (3)
'H' Reflex Latency (milli sec.)	28.464 ± 2.55314	33.468 ± 3.34358	31.372 ± 3.56762
'F' wave Latency (milli sec)	29.523 ± 3.60609	37.868 ± 7.82880	32.45 ± 4.07693
MNCV of Tibial nerve (m/sec)	51.357 ± 3.59956	39.004 ± 7.18295	44.514 ± 6.45477

EMG Study	t^{1-2}	t^{1-3}
	****	****
'H' Reflex Latency	7.1382914	4.0171957
	***	
'F' wave latency	3.4526670	1.9214
	****	****
MNCV of Tibial nerve	9.94960688	5.9719749

n for 'H' reflex (1) 38 (2) 33 (3) 34
n for 'F' wave (1) 12 (2) 19 (3) 14
n for MNCV (1) 40 (2) 52 (3) 48

Significant at * - p less than 0.05, ** p less than 0.02, *** p less than 0.01, ***** p less than 0.0001

Table 6

'H' Reflex & 'F' wave latency - control Vs IDDM NIDDM

EMG Study	Control (1)	IDDM (2)	NIDDM (3)
'H' Reflex Latency (milli sec.)	28.464 ± 2.55314	33.546 ± 3.24021	32.127 ± 3.64662
'F' wave Latency (milli sec)	29.523 ± 3.60609	38.500 ± 10.89025	34.193 ± 3.42172
MNCV of Tibial nerve (m/sec)	51.357 ± 3.59956	39.659 ± 6.63575	42.443 ± 7.58357

EMG Study	t^{1-2}	t^{1-3}
'H' Reflex Latency	**** 5.7782159	**** 5.3361034
'F' wave latency	** 2.7034769	**** 3.7332835
MNCV of Tibial nerve	**** 9.1283503	**** 7.58357

n for 'H' reflex (1) 38 (2) 13 (3) 54
n for 'F' wave (1) 12 (2) 11 (3) 22
n for MNCV (1) 40 (2) 24 (3) 76

Significant at * - p less than 0.05, ** p less than 0.02, *** p less than 0.01, ***** p less than 0.0001

Table 7

'H' Reflex & 'F' wave latency -
Control Vs Newly diagnosed Diabetics

EMG Study	Control (1)	Newly diagnosed (2)	diabetics t^{1-2} (3)
'H' Reflex Latency (milli sec.)	28.464 ± 2.55314	29.900 ± 3.16012	1.6036599
'F' wave Latency (milli sec)	29.523 ± 3.060809	35.850 ± 4.32262	2.9061875 ****
MNCV of Tibial nerve (m/sec)	51.357 ± 3.59956	43.939 ± 6.39179	5.5113811

n for 'H' reflex (1) 38 (2) 12
n for 'F' wave (1) 12 (2) 4
n for MNCV (1) 40 (2) 16

Significant at * - p less than 0.05, ** p less than 0.02, *** p less than 0.01, ***** p less than 0.0001

Table 8

Correlation of severity of Diabetes
Motor nerve conduction velocity

Types of Diabetes	Ulnar nerve Proximal	Ulnar nerve Distal	Median nerve Proximal	Median nerve Distal	Tibial nerve	Peroneal nerve
IDDM	-0.017 *	0.041 ***	0.004	-0.039	0.209	0.461
NIDDM	- 0.362	-0.482	0.197	-0.005	-0.039	-0.235

Significant at * - p less than 0.05, ** p less than 0.02, *** p less than 0.01, ***** - p less than 0.0001.

Table 9

Correlation of duration of Diabetes
Motor nerve conduction velocity

Types of Diabetes	Ulnar nerve Proximal	Ulnar nerve Distal	Median nerve Proximal	Median nerve Distal	Tibial nerve	Peroneal nerve
IDDM	-0.050	-0.048	-0.159	-0.197	-0.173	-0.027
NIDDM	0.132	-0.158	-0.328	-0.086	-0.074	-0.136

p value for Pearson's correlation coefficient

None of the above reaches statistically significant level

Table 10

EMG parameters Vs Age, Duration and Severity of Diabetes
Pearson's Correlation Coefficient

Diabetic status	'H' reflex latency n = 35	'F' wave latency n = 18	MNCV in meters/second n = 50
Age	0.0525798	-0.3150161	0.0406479
Duration	0.0407047 ***	- 0.1765840 *	- 0.0320639
Severity	0.4354969	0.4280908	-0.1698256

Significant at * - p less than 0.10, ** p less than 0.05, *** p less than 0.02.

years of age, distribution of cases showed patients also between 10 years to 50 years of age. Motor nerve conduction velocity of all the peripheral nerves viz., median, ulnar, peroneal and tibial nerve and "H" reflex latency study explored subclinical neuropathy with statistically significant delay in motor nerve conduction and "H" relfex latency. The delay in motor nerve conduction velocity was significant greater in the distal segment of median and ulnar nerve than in the proximal segment. Severe delay in motor nerve conduction velocity was observed in lower limb than upper limb. IDDM patients exhibited statistically significant reduction in motor nerve conduction velocity in the proximal segment of ulnar nerve and distal segment of median nerve than NIDDM. However IDDM patients did not show statisfically significant delay in "H" reflex & "F" wave latencies. Evaluation of terminal latency in all the peripheral nerves showed statistically significant dely in latency except in ulnar nerve in the group without neuropathy. Newly diagnosed diabetics also showed significant delay in motor nerve conduction, "H" reflex and "F" wave latency except terminal latency of tibial nerve. The motor nerve conduction velocity did not evince correlation with severity of diabetes. Where as "H" reflex and "F" wave latency evinced statistically significant correlation with severity of diabetes. However all the three parameters viz., motornerve conduction velocity; "H" reflex and "F" wave latency did not show any correlation with duration of diabetes. In the study by Vijayan etal, significant delay was observed in motor nerve conduction velocity in the distal segment than proximal segment of upper limb as well as greater reduction in motor nerve conduction velocity in lower limb than upper limb. They also explored no correlation with duration of diabetes mellitus and however obtained correlation with severity of diabetes[8]. Stephan C and associates have also observed that there was no correlation between motor nerve conduction velocity and duration & severity of diabetes millitus[9]. Lamontagne and Buchthal described reduction in motor nerve conduction velocity in neuropathic group only[10]. JD Ward etal also identified reduction in motor nerve conduction velocity in newly diagnosed diabetics[11]. Comi G etal also showed electrophysiogical abnormality of peripheral nerves in newly diagnosed diabetic children[12].

Conclusion

This study emphasizes the following inferences : (1) subclinical neuropathy in diabetes mellitus can be identified by motor nerve conduction velocity of all the peripheral nerves, and "H" reflex & "F" wave latency, (2) the delay in motor nerve conduction was much pronouced in distal segment than proximal segment of ulnar and median nerves, (3) the delay in motor conduction was greater in lower limbs than upper limbs, (4) IDDM patients exhibited statistically significant reduction in motor conduction velocity in the proximal segment of ulnar nerve and distal segment of median nerve than NIDDM, (5) newly diagnosed diabetics showed significant delay in motor nerve conduction of all the peripheral nerves except terminal latency of tibial nerve, and "H" reflex & "F" wave lateney, (6) "H" reflex & "F" wave latency evinced good correlation with severity of diabetes.

References

1. Sadikot S.M. Antioxidant vitamins, traditional cooking fats identified as key to diabetes prevention, Medical Times 1993; XXIII (12) : 1-5.
2. Aiello I etal. Diabetic polyneuropathy and insulin secretion in type II diabetic patients, Eur Neurol 1987; 27 (4) : 251 -7.
3. Young R J etal. Progression of subclinical polyneuropathy in young patients with type I (insulin - dependent) diabetes: association with glycemic control and microangiopathy (microvascular complication), Diabetologia 1986 Mar; 29(3): 156 - 61
4. Beccaria L etal. Peripheral nerve abnormalities in newly diagnosed diabetic children, Acta Diabetol Lat 1986 Jan - Mar; 23(1) : 69-75.
5. Sir stanley Davidson and John Aacleod. Diabetes mellitus. In: The principles and practice of Medicine, 10th edition, Great Britain; The English Language Book Society and Churchill Livingstone 1971; 786 - 837.
6. Joseph Good gold. Motor and sensory nerve conduction measurements. In : Electrodiagnosis of Neuromuscular Disease, 2nd Edition, Baltimore : Williams & Wilkins company; 98-104.
7. Williams, Brown. The recording of conducted electrical potentials in nerve trunks and conduction in human motor and sensory fibres. In: The physioligcal and technical basis of electromyography, Boston, USA: Worth Publisher 1984; 145 - 148.
8. Vijayan, Nannihal Singh, Subimal Roy, Pathak. Diabetic neuropathy : a clinical electrophysiological and histological study, Indian Journal of Medical Research 1971 12th December (59): 442 - 452.
9. Stephan C Duck, Herbert T. Nerve function in type I diabetics : Clinical and electrophysiological features, Diabetes 1985; (34) : 115A.
10. Lamontagne A, Buchthal F. Electrophysiological studies in diabetic neuropathy, J Neurology Neurosurgery Psychiatry 1970; 442 - 452.
11. Ward J D etal. Improvement in nerve conduction following treatment in newly diagnosed diabetics, Lancet 1971 27th February; 428-431.
12 Comie G etal Peripheral nerve abnormalities in newly diagnosed diabetic children, Acta Diabetol Lat 1986; 23 (1) : 69 - 75